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The report considers the risk of fatal cancer for the entire fuel cycle due to radiation exposure associated with the annual production of one gigawatt (1000 megawatts) of nuclear electric power. Three sources of radiation exposure are examined: (1) occupational exposure to radiation workers; (2) exposure of the general population from routine releases of radioactivity to the environment; and (3) exposure due to accidental releases of radioactivity. In the analysis, exposures to radiation due to uranium fuel cycle are projected for a period of 100 years following the production of electricity. The major potential impact on human health is from radionuclides intentionally released into the environment during the milling of ores and at fuel reprocessing. It is estimated that about one cancer death could be committed for each gigwatt-year of electrical energy produced.

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ESTIMATES OF THE CANCER RISK DUE TO NUCLEAR-ELECTRIC POWER GENERATION

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PREFACE

This paper was presented at the meeting on "Origins of Cancer," held at the Cold Spring Harbor Laboratory, September 17 through September 14, 1976. The meeting was arranged by J. D. Watson, Director, Cold Spring Laboratory and H. Hiatt, Dean, Harvard University School of Public Health. The proceedings of the meeting will be published in 1977 as the fourth volume in the Cold Spring Harbor Cell Proliferation Series.

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Introduction

Exposure to ionizing radiation results in an increased risk of cancer. The degree of risk incurred per unit dose can be determined, however, only from data obtained mainly at high doses and dose rates; therefore it is necessary to assume a theoretical dose response relationship to estimate the degree of risk from small doses of radiation. The most generally accepted procedure is to estimate cancer risk on the basis of a linear non-threshold hypothesis; i.e., to assume that the number of cancers which may occur per unit dose at low doses and dose rates is the same as observed at high doses and dose rates. Following this practice, we have used the radiation risk estimates for fatal cancer given in the 1972 "BEIR" report by the National Academy of Science (NAS 1972). In this analysis, we have considered the risk of fatal cancer for the entire fuel cycle due to the radiation exposure associated with the annual production of one gigawatt (1000 megawatts) of nuclear electric power. A gigawatt-year (GW(e)-y) is the amount of energy produced annually by a single nuclear reactor of the type and size currently being constructed.

Three sources of radiation exposure are examined: occupational exposure to radiation workers, exposure of the general population from routine releases of radioactivity to the environment and, finally, exposure due to accidental releases of radioactivity. The quality of data available for risk assessment

varies depending on the source of exposure being considered; good for routine releases, fair for occupational exposure and relatively poor for accidents.

Cancer risks due to nuclear electric power generation are somewhat unique in that the release of some of the radioactive materials implies a dose commitment to future generations which persists over such long periods of time (millenia) that the risk cannot be quantified meaningfully. If, however, the assessment is confined to health effects expected to occur within a few generations, usable estimates of the number of fatal cancers per gigawatt-year can be made. In this analysis, exposures to radiation due to the uranium fuel cycle are projected for a period of 100 years following the production of the electricity. The impact over longer time periods is discussed only qualitatively.

Routine effluent releases from nuclear reactors are not the focal point of this assessment since, except for occupational exposures, they are relatively insignificant sources of radioactive pollutants. Rather, the risk assessment should include the entire uranium fuel cycle, which involves the mining and processing of uranium ores, uranium enrichment, fuel fabrication, fuel reprocessing, and finally waste storage and disposal. Unfortunately, options for the disposal of radioactive materials following fuel reprocessing are as yet poorly developed

and the environmental risk from uranium mining has received little attention. Therefore, the risks to the general population associated with waste disposal and mining are not accounted for in this analysis.

Technical Considerations

The sequence of operations occurring before and after the fissioning of fuel at the power reactor is shown schematically in Figure 1. Uranium ore, which usually contains approximately 0.2% uranium, is first mined and then milled to produce a concentrate called "yellowcake" which is about 75 percent uranium. The greatest portion of the feed ore becomes a plant residual depleted in uranium but containing radium-226 and its long half-life parent thorium-230. These "tailings" are a source of radon-222 emissions which can last for several hundred thousand years.

Following the manufacture of yellowcake, a conversion step purifies and converts the uranium oxide to uranium hexafloride, the chemical form in which uranium is supplied to an enrichment plant. At this plant, the isotopic concentration of uranium-235 is increased from its natural abundance, about 0.7% uranium, to the design specification of the power reactor, usually 2-4%, by a differential gaseous diffusion process. The enriched uranium hexafloride is converted into uranium oxide pellets at a fuel fabrication plant and then loaded into thin zircalloy or

stainless-steel tubes which are fabricated into fuel element bundles.

Current plans call for the reprocessing of spent fuel after a three-year "burnup" in the reactor. At this point the primary physical barriers to the release of radioactivity are removed and residual gaseous fission products, mainly tritium and krypton-85, are released into the environment. Control of krypton-85 effluents is technically feasible and is the subject of a current Federal rulemaking. Most of the remaining radioactive materials, with the notable exception of carbon-14, are recovered and either recycled or stored permanently as indicated in Figure 1.

Environmental Dose Commitment

Planned releases of radioactive materials occur in every step of the uranium fuel cycle. However, the major potential impact on human health is from radionuclides intentionally released into the environment during the milling of ores and at fuel reprocessing. The importance of ore milling and fuel reprocessing to health considerations is not always recognized since most risk assessments are confined to a consideration of the annual dose rate delivered to persons living in the vicinity of the release point. Such temporally and spatially limited studies do not address adequately the dose committed from the discharge of long half-life radioactive materials into the biosphere. Krypton-85 released from a fuel reprocessing plant is

a case in point. Given an adequate stack height for the release of this noble gas, individual annual doses are not large in terms of currently permitted annual dose levels. However, its relatively long half-life (10.7 years) allows world-wide atmospheric diffusion and, since there are no environmental sinks, krypton-85 persists in the atmosphere for several decades. In other words, the production of a given amount of nuclear electricity commits to the environment a material which will deliver an impact in subsequent years due to the persistence of radioactive materials. The total dose considered from this perspective is termed the "environmental dose commitment" and, simply defined, is the sum of the doses to all individuals over the entire time period the material persists in the environment in a state available for interaction with humans (Richardson 1974).

Health Effects from Planned Releases

Health effects due to 100-year environmental dose commitments from the uranium fuel cycle have been considered in a number of recent EPA studies published in support of proposed Federal environmental radiation standards, as cited below. These analyses are based on the expected performance of average facilities operating under current control requirements. They do not take into account the effect of any additional control measures which may be instituted in the future.

Table 1 indicates the estimated number of fatal cancers due to the 100-year environmental dose commitment for that portion of the fuel cycle that preceeds the generation of electrical energy (EPA 1973-1). Clearly, of all the sources considered, radon emissions from tailings piles dominate the risk estimate. This is due to several factors that are often not appreciated. Typically, the duration of transcontinental transport in the troposphere ranges from four to eight days. Therefore, the 3.8 day half-life of radon is not too short to allow the widespread diffusion of this radionuclide into populated areas. Studies by the Environmental Protection Agency indicate a factor of about 0.4 for radon decay between points of emission in the Rocky Mountain states and the eastern seaboard of the United States. Atmospheric transport beyond the Atlantic Coast has not yet been studied in detail. Our preliminary estimate indicates that about half of the projected cancers due to these emissions would be expected to occur in the U.S., the balance occurring in the rest of the northern hemisphere.

Table 1
Estimated Cancer Fatalities due to the Extraction and Fabrication of Uranium Fuel
(100-year environmental dose commitment)

Industry	Principal Radionuclides	Fatal Cancers per GW(e)-y
Uranium Milling	radon-222	5×10^{-1}
UF_6 Conversion	uranium	1×10^{-2}
UF_6 Enrichment	uranium	1×10^{-3}
Fuel Fabrication	uranium	6×10^{-5}
Transportation	uranium	2×10^{-4}
		$\Sigma = 0.5$

The duration of radon emission from tailings piles is not controlled by its parent isotope radium-226, which has a 1602 year half-life. A 1600 year half-life could at least be dealt with conceptually; but, unfortunately, the radium-226 in tailings piles is in near equilibirum with its parent, thorium-230, which has an 80,000 year half-life. Granted that a 100-year dose commitment seems particularly inapplicable to this case, it seems equally unwise to predict that a tailings pile will continue to emit radon into the biosphere over a time period so long that geological stability cannot be assured. This brings to mind a third point. Some investigators argue that since the radon emissions are from a naturally occurring radioactive material, the health effects would occur anyway and, therefore, should not be charged to the nuclear fuel cycle (Cohen 1976). Such a

viewpoint fails to recognize that without man's intervention this radon would decay deep within the earth and not enter the biosphere. Under these circumstances it would seem reasonable not to differentiate between the release of radon from tailings piles and the release of krypton-85 during fuel reprocessing. It can be seen from Table 1, that compared to the potential health impact due to radon, planned releases from other portions of this part of the fuel cycle have much lesser impact.

Estimated numbers of fatal cancers from the 100-year environmental dose commitment due to the reprocessing of the spent fuel which has produced one gigawatt-year of electrical energy are shown in Table 2 (EPA 1973-2). Until fairly recently it had been assumed that krypton-85 and tritium were the most important radionuclides involved. However, Magno and coworkers have pointed out, rather surprisingly, that the dose commitment from carbon-14 exceeds that from all other radionuclides combined (Magno, Nelson and Ellett 1974). Carbon-14 is not a fission product. It is produced via an n,p reaction on nitrogen-14 impurities in uranium fuels and to lesser extent from an n, reaction on oxygen-17 in reactor cooling water (Fowler, et. al., 1976).

Table 2
Estimated Cancer Fatalities due to the Reprocessing of Spent Fuel
(100-year environmental dose commitment)

Principal Radionuclides	Fatal Cancers per GW(e)-y
Carbon-14	2.3×10^{-1}
Tritium	1.8×10^{-2}
Krypton-85	4.6×10^{-2}
	$\Sigma = 0.3$

Carbon-14 dioxide injected into the atmosphere during fuel reprocessing moves from inorganic reservoirs to living systems. The concentrations of this carbon-14 in man has been calculated using a world transport model for carbon-14 developed by Machta (Machta 1973). This model is a multi-reservoir exchange model consisting of seven compartments with exchange rates based on the analysis of carbon-14 produced in nuclear weapons testing. The model takes into consideration projected increased levels of tropospheric carbon due to the combustion of fossil fuels which are depleted in carbon-14. The 100-year environmental dose commitment from carbon-14 has been calculated on the basis of its specific activity in tissue. This assumes an equilibrium between tissue and the troposphere and may underestimate individual doses slightly in the vicinity of a fuel reprocessing facility.

The estimated number of fatal cancers due to the 100-year environmental dose commitment from a reactor producing one gigawatt-year of electrical energy are listed in Table 3 (EPA

1973-3, EPA 1976-2, Fowler, et. al., 1976). Except for carbon-14, these dose commitments are negligible compared to these from the balance of the fuel cycle.

Table 3
Estimated Fatal Cancers due to Nuclear Reactor Effluents
(100-year environmental dose commitment)

Principal Radionuclide	Fatal Cancers per GW(e)-y
Carbon-14	9×10^{-2}
Krypton-85	7×10^{-5}
Tritium	8×10^{-5}
Short half life effluents	$\frac{<1 \times 10^{-2}}{\Sigma = 10^{-1}}$

Occupational Exposures

Contrary to the case for exposures from planned effluent releases, occupational doses due to nuclear energy production are dominated by the exposure of nuclear reactor operators and maintenance personnel. Recently, Golden and Pavlick have reviewed occupational doses as a function of plant age at the two largest and oldest multiple reactor complexes in the United States, Quad Cities and Dresden (Golden and Pavlick 1974, Golden 1976). These studies show that the early operating experience at a reactor is not indicative of later occupational doses. Occupational exposure increases with plant use as a result of the activation of structural materials and equipment as well as increased inventories of radioactive materials in storage areas.

Occupational exposure also occurs more frequently due to the need for increased plant maintenance with plant age. Indeed, maintenance operations, both scheduled and unplanned, lead to about half the total occupational dose and impact mainly on persons, such as welders, not routinely employed in the operation of reactors. The mean annual occupational dose per reactor at Dresden, the oldest of these reactor complexes is 1050 man-rem and at Quad city 800 man-rem. Even though these reactors produce less than a gigawatt year annually, occupational doses are not a sensitive function of the reactor's generating capacity and a rather conservative estimate of the population dose due to occupational exposure is judged to be about 800 man-rem per gigawatt-year (electric) as shown in Table 4. Nuclear Regulatory Commission (NRC) estimates for occupational exposures at nuclear power reactors and for the balance of the fuel cycle are also included in Table 4 (AEC 1974).

Table 4
Estimated Cancer Fatalities due to Occupational Exposure

Industry	Total Body Man-rem EPA	NRC	Fatal Cancers per GW(e)-y
Mining	---	---	2×10^{-2}
Reactors	800	(235)	1.5×10^{-1}
Fuel Fabrication	---	40	
Fuel Reprocessing	---	12	$1\text{-}2 \times 10^{-2}$
Transportation	---	0.5	
Waste Management	---	2	
			$\Sigma = 0.2$

The dose to uranium miners, the only class of workers in the fuel cycle for which there is epidemiological evidence of radiogenic cancer, has not usually been included in occupational risk estimates, since underground miners are not considered to be radiation workers under the Atomic Energy Act. Radon exposures to uranium miners are controlled by the States, under Federal guidance. Current limits for radon daughters (^{218}Po, ^{214}Pb, ^{214}Po) are expressed in terms of cumulative working levels. In a single year an underground uranium miner may be exposed to four working level months (WLM). Average exposures probably range between one and three WLM annually (MESA 1975). On the basis that 53 man-years of mining effort are required to obtain the ore necessary to produce one gigawatt-year (electric), the occupationally induced

lung cancer death for the ore needed to generate one gigawatt-year of electricity is estimated as about 0.02.* Though this last calculation is imprecise, due to the lack of reliable data on exposure to miners, it is probably realistic to assume that the nuclear reactor is the most important source of occupational exposure in the nuclear energy industry.

Accidents as a source of population dose commitment

While unplanned releases of radioactivity into the biosphere as a result of accidents at uranium fuel cycle facilities are a potential source of radiation-induced cancers, little can be said with much confidence about their likelihood or the effects of such accidents on the general population. Only for nuclear reactors has a significant start been made towards predicting this risk. Two factors are involved in such analyses: a consequence model, which yields the distribution of doses that would occur on the average from a postulated accident, and a fault tree analysis that yields the joint probability that a given sequence of events will actually occur, given the probability of individual component failure, power failure, etc. In the final analysis these probabilities are essentially engineering judgments. Therefore, such risk assessments are dependent on "Bayesian estimates" reflecting degrees of faith in particular engineering practices rather than on "Fisher likelihood estimates," which draw on an actual sample of observed

*It is assumed that two-thirds of the uranium ore is obtained by underground mining.

events. Not suprisingly, knowledgeable individuals have different degrees of faith based on their own prejudgments.

The most serious reactor accidents are those involving meltdown of the reactor core where containment is more or less lost and substantial quantities of some of the reactor contents enter the environment. In addition, there are non-core accidents where the quantity of material released is generally much smaller. The recently published Reactor Safety Study, more commonly known as the "Rassmussen Report," evaluates numerically the population dose due to core related accidents (NRC 1975). The cancer risks reported in the Reactor Safety Study are not directly comparable to those given in this analysis since the estimated number of cancers per unit population dose utilized in that Study is approximately a factor of four smaller than those given in the NAS BEIR Report (EPA 1976-1). Table 5 lists the projected number of cancers per gigawatt year (electric) from large reactor accidents based on the Reactor Safety Study, after taking this factor of four into account.

Table 5
Estimated Cancer Fatalities from Accidental Release of Nuclear Material

Source	Fatal Cancers per GW(e)-y
Nuclear Reactor (core failure)	8.4×10^{-1}
Nuclear Reactor (non-core related)	unknown
Fuel Reprocessing	unknown
Transportation	10^{-7} (EPA 1975)
Waste Storage	unknown
	$\Sigma \simeq .1(?)$

Major accidents in the balance of the fuel cycle should have a much smaller impact on the general population. For example, the amount of short half-life gaseous radioactivity involved at any one time at a fuel reprocessing operation is much less than in the case of an operating reactor. There remains the question of the health impact from less serious reactor accidents. To date only individual doses, not the population dose commitment, have been examined for this class of accidents. These lesser accidents are of some interest, since their estimated likelihood of occurrence is about one hundred times greater than that for accidents involving core meltdown.

Current regulations for identified non-core related accidents impose design guidelines that will limit individual exposure at the facility boundaries to doses ranging from 500 to 2000 mrem. Weighting these doses by their expected frequency of

occurrence yields a total body dose of 35 mrem per gigawatt-year (electric) to the most highly exposed individual in the general population. This is about 10 to 20 times larger than the maximum total body dose of 2-4 millirem per year expected from routine releases (EPA 1976-2). Even though maximum individual doses are not directly proportional to the total dose commitment to populations, it would appear to be worthwhile to evaluate the 100 year environmental dose commitment caused by minor reactor accidents. Table 5 is an admission that, in reality, very little is known about the potential dose commitment and health effects due to accidents in the uranium fuel cycle. Until recently, accident analysis for the licensing of nuclear facilities has been exclusively concerned with consequences in the immediate vicinity of the facility so that appropriate emergency plans may be implemented if needed. There is a great deal of interest in expanding this limited viewpoint, and it is reasonable to expect significant changes in the amount of information that will be available within the next few years.

Adequacy of NAS-BEIR estimates of radiocarcinogenesis

Before considering the total potential health impact from one gigawatt-year of nuclear electric energy production it is worthwhile to consider the degree of confidence that can be placed in current estimates of the cancer risk due to radiation. Although there is general agreement in both the United States and

in international scientific communities that the NAS-BEIR report is a comprehensive analysis of the radiation effects data for human exposures available in 1972, it is being argued by some that in their calculation of the cancer risks from ionizing radiation, the BEIR Committee did not give sufficient weight to animal data on radiation carcinogenesis. More specifically, this criticism is based on the observation that the incidence of some radiation induced cancers in rodents is reduced at low doses (and/or low dose rates) of low LET* radiation. The hypothesis is that repair of precancerous damage reduced the cancer incidence. The revisionists' contention is that this postulated repair of radiation damage should be taken into account when estimating the risks from nuclear energy. While there is little or no evidence for such repair in large mammals or in man, it has some theoretical basis in the model of radiation injury proposed by Kellerer and Rossi (Kellerer and Rossi 1971). Their theory of dual radiation injury holds that initial radiation injury, i.e., damage to critical sites, has both linear and quadratic dependence on dose.

$$y = a D + b D^2$$

Where the coefficients a and b correspond to one and two "hit" damage respectively. For high LET radiations the first coefficient, a, is so much greater than the second, b, that the dose squared term can be neglected at all dose levels. This is

*Lightly Ionizing.

not true for low LET radiations, for which the relative importance of each term depends on the dose range of interest. A quadratic component in the response to radiation has been observed for genetic damage from ionizing radiation and, if one step in the development of a cancer is defined as a somatic mutation, reasonable arguments can be made in support of the applicability of this hypothesis to the initiation of radiocarcinogenesis.

Rather than discuss the theory in critical detail, the point to be made here is that its general acceptance would not have much effect on the conclusions reached in the NAS-BEIR report concerning cancer risk from ionizing radiation. The Rossi-Kellerer theory predicts a linear response for low doses and a supra-linear response at high doses, the dose squared term dominating at doses that exceed the ratio a/b. Obviously, if the human effects data used by the NAS-BEIR Committee were obtained only at high doses, a linear approximation to the dose response data would greatly overestimate the risk. This has been suggested by several groups, including the National Council on Radiation Protection and the Nuclear Regulatory Commission (NCRP 1975, NRC 1975). More recently, a concensus has developed that if the NAS-BEIR risk coefficients do indeed overestimate carcinogenesis, it would be only by a factor of two, an amount comparable to other uncertainties in the application of these

data (Brown 1976, Upton 1976, EPA 1976-2). The basis for this consensus is that current information on the values of a and b (based on genetic studies) indicate that the coefficient a is about 100 times greater than b so that the linear term dominates the response function at doses less than 100 rad or so. This is about the low end of the range of doses considered by the NAS-BEIR Committee. Since, in general, the Committee interpolated linearly between zero and the lowest dose level where excess cancer was observed, it is probably unlikely that their conclusions were heavily biased by a dose response dominated by higher power terms.

Indeed, it is by no means clear that any modification need be made to NAS-BEIR risk estimates. There are many points in favor of using a linear assumption for cancer risk prediction. One is the lack of a supra-linear response in much of the available human data. Figure 2, which is taken from the BEIR Report, indicates human breast cancer increases in a linear manner for a range of doses between 50 to 7000 rads (NAS 1972). Even though this example is not as clear cut as one would like (the higher doses were accompanied by increased fractionization) it seems suprising, in view of the Kellerer-Rossi theory, that so little departure from linearity is observed at doses as large as 7000 rad.

Turning to the other end of the scale, the low dose range, again the BEIR risk estimates seem to be holding up rather well. The 1972 BEIR estimates of the radiation risk per rem for thyroid cancer in children ranged from 1.6 to 9.3 cancers per 10^6 persons years at risk (NAS 1972). These results were obtained from a variety of epidemiological series in which the average thyroid dose exceeded 200 rem. More recently, Modan and coworkers (Modan, et. al., 1974) have reported on a follow-up study of 11,000 Israeli children treated with x-rays for tinea capitis, a fungal scalp infection. Because a great deal of care was exercised in beam direction and in the use of patient sheilding, the estimated thyroid doses, compared to those considered by the BEIR Committee, were quite low, varying from 6.2 to 7.4 rem with a mean of 6.5 rem. The observed excess thyroid cancer incidence per rem, using siblings as controls, was 6.9 per 10^6 person years at risk. This finding is in agreement with the upper range of the NAS-BEIR estimate, cited above, and does not support a hypothesized reduced response at low doses. Rather than argue that thyroid and breast cancer are unrepresentative of radiogenic cancers in man, and therefore exceptions to an apparent recovery from precancerous radiation damage observed in rodents, it would appear more prudent and indeed advisable to limit exceptions to the NAS-BEIR risk estimates to those human cancers where supporting epidemiological data is available.

This is not to say the NAS-BEIR estimates are precise. They are not. Not only is the human data on which they are based quite limited, but there are also critical questions as to how the BEIR risk estimates should be applied to heterogeneous population groups, since they are based, in large part, on adult data. The NAS-BEIR Report provides two estimates of radiation risk. One is an estimate of the absolute risk, that is the number of cancers that will occur per unit dose in an exposed population. The other estimate is of relative risk, the percentage increase in the excess cancer mortality per unit dose. When lifetime exposure to a population containing a mixed age distribution is considered, the number of fatal cancers per person rem estimated on the basis of relative risk exceeds the number estimated on the basis of absolute risk by a factor of about four. In the previous analysis, averages of the relative and absolute risk were used to predict the cancer risk from nuclear generated electricity. This procedure will underestimate the actual risk if radiation-induced carcinogenesis increases in proportion to the cancer incidence in an unirradiated population. There is some limited data on this point from the Japanese atomic bomb casualties. Figure 3, taken from the NAS-BEIR Report, illustrates the difference between these two models (NAS 1972). The two curves compare the age-specific cancer death rate in a

Japanese control group with the age specific absolute risk for fatal solid tumors in atomic bomb survivors.

If the absolute risk model is an accurate predictor of cancer mortality from atomic bomb radiation, the incidence of radiation cancers would be independent of age at exposure, i.e. it would appear as a horizontal line parallel to the abcissa. On the other hand, if the risk increases in proportion to the age-dependent incidence of 'spontaneous' tumors, the observed age-specific risk from bomb exposures would parallel the age-specific mortality rate of the control population. As can be seen, the correlation between radiation induced and non-radiogenic cancer is high, particularly if the increased sensitivity of children to radiation is taken into account. One should note, however, the large uncertainty associated with radiation cancer mortality for the 0 to 9 age group in Figure 3. The fact is that as yet there have been very few deaths due to solid tumors in this age group, only nine for exposures greater than 100 rads. As this group ages* it will be of interest to observe to what extent their cancer frequency reflects that of the general Japanese population and their radiation exposure history. Perhaps in twenty years or so we will be able to exclude eitner the relative or absolute risk model, for the most common cancers.

In view of the emphasis that is placed on the Japanese studies in some assessments of radiation risk, the uncertainties

*The persons at risk are between 31 and 41 years old now.

associated with these data should be more widely appreciated, particularly the unreliability of these data for elucidating the form of the dose response function for low LET radiation. Recently, a number of investigators (Mays, Lloyd and Marshall 1975; Rossi and Kellerer 1974; Jablon 1974) have suggested that the incidence of leukemia observed after the atomic bombing of Nagasaki can be used to assess the risk from low LET radiation since, unlike the situation at Hiroshima, the neutron dose to Nagasaki survivors is negligible. A tentative conclusion that has been drawn from such studies is that the incidence of leukemia varies as the square of the dose for low LET radiations rather than linearly.

An important question that has not been considered explicitly in any of these studies is whether or not the leukemia data obtained on Nagasaki survivors is sufficient to permit any meaningful discussion of the dose-response relationship at doses below 100 rad or so, since the number of persons at risk in the Nagasaki study population is relatively small compared to that from Hiroshima. Table 6 compares the number of leukemia cases reported in the Nagasaki Cohort Study with that expected from Japanese national death rates published by the Japanese Ministry of Health and Welfare. All of the data in Table 6 is taken from ABCC Report 10-71 (Jablon and Kato 1972).

Table 6
Observed & Expected Leukemia Mortality in Nagasaki Survivors[1]

Mortality	Mean dose (rads) 2.4 (0-9)	21.3 (10-49)	70.3 (50-100)	144.3 (100-200)	329 (200-600)
Observed (O)	11	2	0	3	15
Expected (E) no radiation	5.6	1.8	0.6	0.6	0.7
Excess (O-E)	5.4	0.2	-.6	2.4	14.3

[1]Cohort Study, Jablon and Kato 1972.

From Table 6 it is seen that when the expected "background" cancer incidence is subtracted from the observed, there is no dose response data for total body doses below 100 rad*. It is apparent that the net number of excess effects observed precludes a realistic differentation between linear and dose squared response patterns in the dose range of interest.

The paucity of effects information shown in Table 6 does give rise to at least one more question. In view of the lack of excess cancer below 100 rad, is the Nagasaki leukemia data consistent with risk estimates given in the BEIR Report, which are based on an assumed linear nonthreshold dose response relationship? BEIR risk estimates for leukemia separate the irradiated populations into two risk groups; those over ten years of age, and survivors who were less than ten at the time of the bombing. The BEIR absolute risk estimate (based on all leukemia results and not just the Japanese studies) assume for adults one

*Use of other control groups to estimate the exposed mortality, such as persons not in the city at the time of the bombing, does not alter this finding.

case per rad per 10^6 person years at risk and for children, twice this number of cancers per rad (NAS 1972). Tables 7 and 8 compare the leukemia incidence in the Nagasaki Cohort Study for the older and younger age groups (Jablon and Kato 1972) with that predicted using the risk coefficients given in the BEIR Report. They indicate that for doses less than 200 rads the net leukemia mortality among Nagasaki survivors is about what would be expected on the basis of BEIR estimates. Certainly, the Nagasaki data does not appear to provide an adequate basis for rejecting a linear dose response relationship.*

Table 7
Nagasaki Leukemia Mortality in Persons Over 10 Years of Age at Time of Bomb[1]

Mortality	Mean dose (rads) 2.4 (0-9)	21.3 (10-49)	70.3 (50-100)	144.3 (100-200)	329 (200-600)
Persons at risk	5158	3719	983	1030	1110
Observed(O)	8	2	0	2	8
Expected(E)	4.5	1.4	.5	.5	.6
Excess (O-E)	3.5	.6	-.5	1.5	7.4
BEIR Estimate	.3	1.0	1.0	2.7	9.8

[1]Cohort Study, Jablon and Kato 1972.

*Note added in proof (second printing)

At the recent 1976 Radiation Research Society Meeting, Beebe, Kato and Land pointed out that when all leukemia fatalities in the Nagasaki Register are considered, and not just those in the ABCC Cohort Study subset, the incidence of leukemia increases monotonically with dose at all dose levels. Although these data are still insufficient to determine a functional relationship between leukemia incidence and dose, they do illustrate the absence of excess leukemia deaths shown in Table 6 is a statistical artifact inherent in the small number of cases recorded in the Cohort Study.

Table 8
Nagasaki Leukemia Mortality in Persons Under 10 Years of Age at Time of Bomb[1]

Mortality	Mean dose (rads) 2.4 (0-9)	21.3 (10-49)	70.3 (50-100)	144.3 (100-200)	329 (200-600)
Persons at risk	1592	981	248	199	200
Observed (O)	3	0	0	1	7
Expected (E)	1.1	.4	.1	.1	.1
Excess (O-E)	1.9	-.4	-.1	.9	6.9
BEIR Estimate	.1	.8	.7	1.0	2.6

[1]Cohort Study, Jablon and Kato 1972.

Conclusions

Having estimated the cancer risk from the uranium cycle and then indicated to you some of the issues concerning the degree of uncertainty in such risk estimates, it can be understood that the projected health risk from nuclear power is just that, an estimate. Table 9 sums up the estimated risk of fatal cancer for the various portions of the uranium fuel cycle. If the risk per gigawatt from accidents and waste disposal are as low as seems likely now, about one cancer death could be committed for each gigawatt-year of electrical energy produced.

Table 9
Estimate of the Total Cancer Fatalities for one Gigawatt-year of Electrical Energy

Planned releases	0.9
Occupational Exposure	0.2
Accidents	0.1
	$\Sigma \approx 1.2$

In our opinion, it is beyond the state of the art to compare these risks to the cancer risks from other forms of electrical energy production. We simply do not know the long term health risk from particulates, SO_2 and other chemical emissions which are inherent with the use of fossil fuels to produce electrical energy. Not as a surrogate for such data but merely to give some indication that no source of energy is without risk, Table 10 compares the occupational risk due to accidents in other fuel cycles for equivalent amounts of electrical energy (CEQ 1973). Obviously, most fossil fuels are not appreciably safer and coal is considerably more hazardous. Having said that, it is also fair to point out that if the cancer estimates for the uranium fuel cycle were based on a 1000-year environmental dose commitment, it would shift the balance in favor of coal if only the occupational risks were counted. However, until we know more about the health effects due to the long term environmental pollution caused by other given energy cycles, comparisons of the health risk for various fuel cycles is premature.

Table 10
Occupational Fatalities for Other Energy Sources

Boiler Fuel	Accidental Fatalities per GW(e)-y
Coal	4.3
Oil	0.42
Gas	0.27

It is worthwhile at this point in the discussion to step back and consider not so much what has been stated, but rather that part of the risk analysis which has not been explored. Numerical estimates of risk from radiation are but one aspect of an overall assessment. Equally important are issues not discussed in this paper. These include such ethical questions as whether or not the risks are voluntary, and the degree of correlation between the distribution of benefits and the distribution of risk among the various segments of the population. Consideration must also be given to the extent to which the risks are subject to control and the cost of such controls. Progress is being made in developing a macroscopic viewpoint for risk analysis so that various energy sources can be compared in a consistent manner (Rowe 1977). Nevertheless the achievement of this goal is still ahead of us.

Finally, it is important to recognize that the implications, for the Nation, inherent in the development and widespread use of any energy source transcend public health considerations alone.

Health risk estimates are easily transcribed by the media into a news story and may perhaps be overemphasized for that reason. Less easily imparted is information on how the selection of prime energy sources will impact on other areas of national life. To name but a few, the continued use of imported oil as a prime energy source will influence foreign policy considerations as well as have implications for the continued integrity of the marine environment. Utilization of coal as an energy resource effects the allocation of large areas of land for periods of time that are comparable to that of long half-life nuclear materials. The institutionalization of domestic security procedures and the commitment of a large portion of the Nation's capital expenditures are inherent in the development of a nuclear economy, particularly if it includes plutonium as an energy source. All of these factors concern alternative benefits rather than health risk. They are not amenable to quantification and can be evaluated only partially on technical grounds. It follows that societal and political judgements must provide the actual basis for any final decisions on the acceptability of risk.

From this broader perspective the importance of public health considerations is not diminished, even though it is obviously only one input to a comprehensive decision-making process. Rather, it points out the need for health risk estimates to be as free as possible of bias concerning the

benefits from any given energy source. The balancing of health risks against societal benefits is not an appropriate responsibility for the scientific community, which by training and perhaps by inclination has little special competence for evaluating societal benefits. Technical judgments on risk should be separated from societal and political judgments concerning benefits, so that decisions can be made on the basis of visible preferences. Unfortunately, there appears to be a growing tendency to include societal considerations in the assessment of risks, particularly nuclear risks. While both critics and proponents of nuclear energy have valid viewpoints which transcend health risk considerations, the discussion of this energy source is often colored by risk assessments that are based on preconceptions of either benefit or harm to society. The discussion of nuclear energy would be more enlightened if these other issues were addressed directly and not weighted into the assessment of health risk.

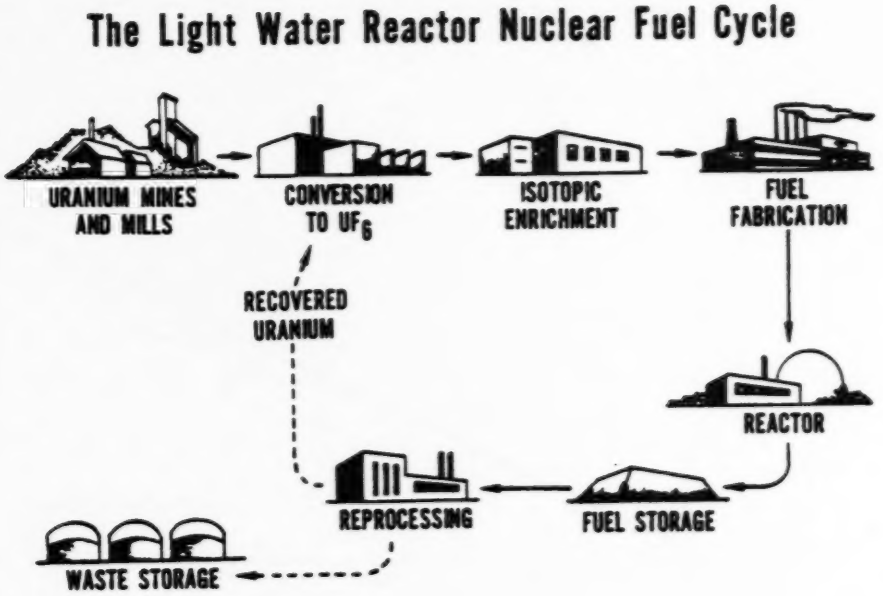

Figure 1: The uranium fuel cycle for light water moderated nuclear power reactors. The material transfers indicated by the dotted lines have yet to be implemented commercially.

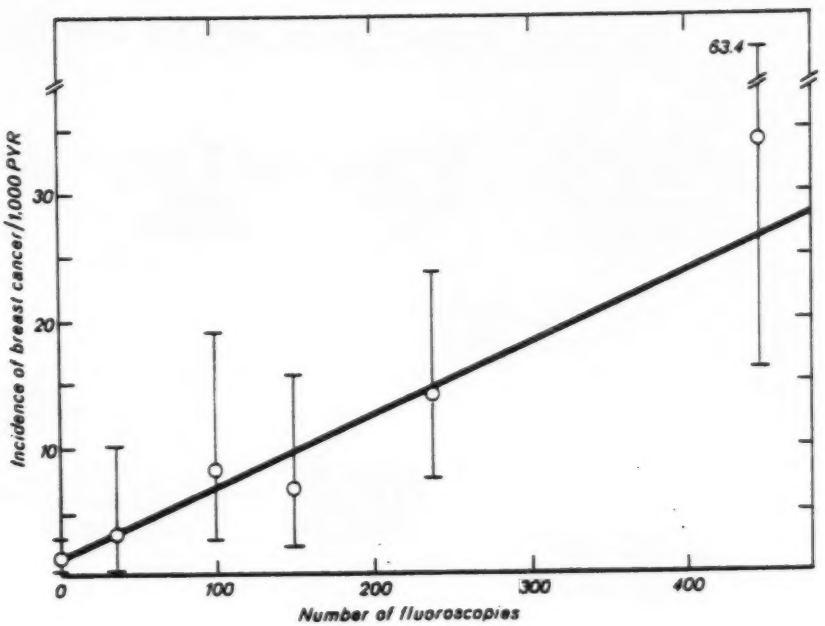

Figure 2: Incidence of breast cancer per thousand person year at risk (PYR). The error bars represent 90% confidence intervals, and the line is the best fitting weighted least squares regression line (NAS 1972).

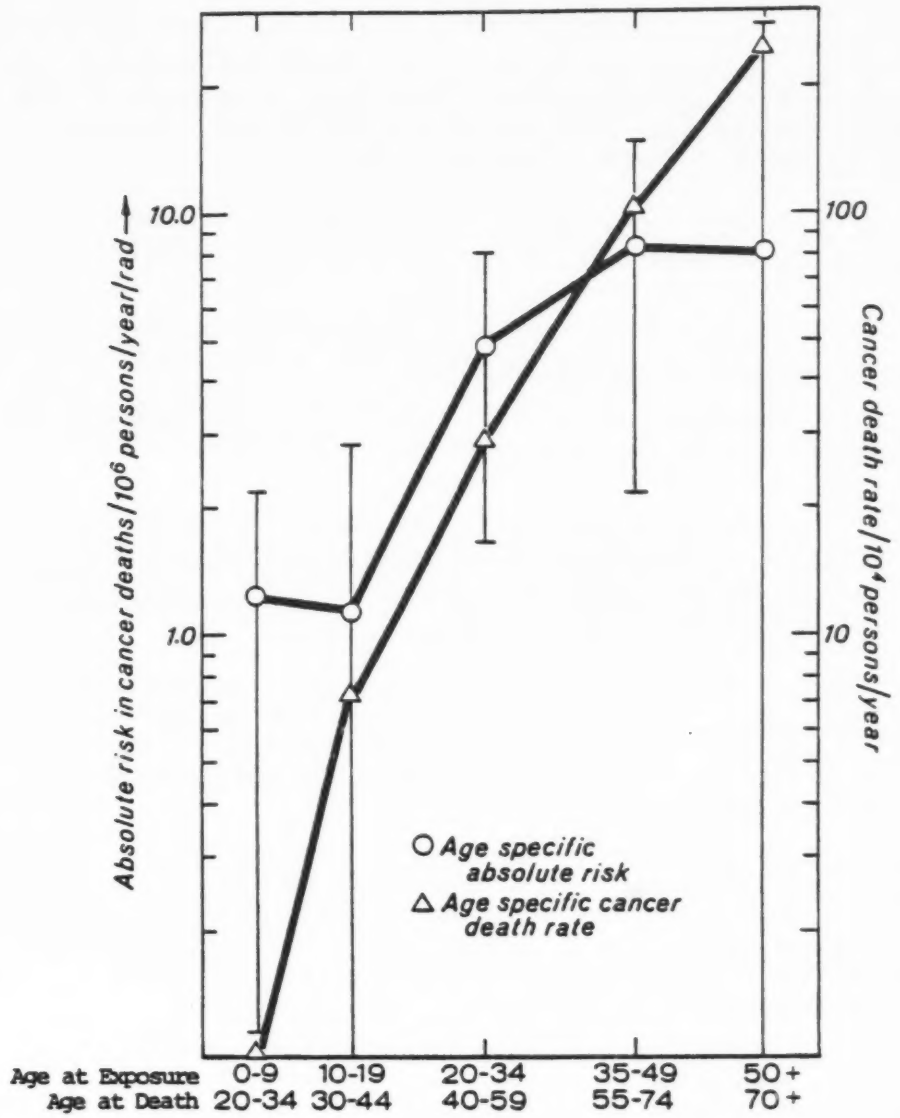

Figure 3: The absolute risk of induction of all malignancies (except leukemia) in the A-bomb survivors versus age. The data are for both cities and sexes combined, for deaths from 1960 to 1970 and no neutron RBE correction has been applied. The age specific death rate has been taken from the death rate in the 0-9 rad control group (NAS 1972).

REFERENCES

Beebe, G. W., H. Kato and C. E. Land, 1976. "Mortality Experience of A-bomb Survivors, 1950-1974, presented at the 24th Annual Scientific Meeting of the Radiation Research Society in San Francisco, June 29, 1976.

Brown, J. M., 1976. Linearity vs Nonlinearity of Dose Response for Radiation Carcinogenesis, Health Physics, Vol. 31, pp. 231-245.

Cohen, B. L. 1976. "Environmental Impacts due to Radon Emissions", Bull. Atomic Scientists, 32, 2, pp. 61-64.

Council on Environmental Quality (CEQ) 1973. Energy and the Environment, pp. 42-53, U.S. Government Printing Office, Washington, D.C. 20402

Fowler, T. W., R. L. Clark, J. M. Gruhlke, and J. L. Russell, 1976. Public Health Considerations of Carbon-14 Discharges from the Light-Water-Cooled Nuclear Power Reactor Industry, Technical Note ORP/TAD-76-3, U.S. Environmental Protection Agency, Washington, D.C. 20460

Golden, J. C., 1976. "Effect of a Change in Occupational Radiation Exposure Limits on Manpower and Total Dose at a Nuclear Power Station". Paper read at the 21st Annual Meeting of the Health Physics Society, Health Physics (Abstract in press).

Golden, J. C. and R. A. Pavlick 1974. "A Review of Effluents, General Population Doses and Occupational Doses Resulting from Commonwealth Edison's Use of Nuclear Power", Symposium on Population Exposures, CONF-741018, pp. 199-206, National Technical Information Services, Springfield, Virginia 22161.

Jablon, S., 1974. "Environmental Factors in Cancer Induction: Appraisal of Epidemiologic Evidence Leukemia, Lymphoma and Radiation," Proceedings of the XI International Cancer Congress, Florence, Excerpta Medica Congress Series No. 351, Amsterdam

Jablon, S. and H. Kato, 1972. "Mortality Among A-Bomb Survivors", Report 6, ABCC TR-01071, Japanese National Institute of Health - Atomic Bomb Casualty Commission Hiroshima and Nagasaki, Japan.

Kellerer, A. M. and H. H. Rossi 1971. "RBE and the Primary Mechanism of Radiation Action", Rad. Res. 47, 15.

Machta, L., 1973. "Prediction of CO_2 in the Atmosphere", Carbon and the Biosphere, CONF-720510, G. M. Woodwell and E. V. Pecan, editors, pp. 21-31, National Technical Information Services, Springfield, Virginia 22161.

Magno, P. J., C. B. Nelson and W. H. Ellett, 1974. A Consideration of the Significance of Carbon-14 Discharges in the Nuclear Power Industry, Proceedings of the 13th AEC Air Cleaning Conference, CONF-740807, Vol. 2, pp. 1047-1055, National Technical Information Services, Springfield, Virginia

Mays, C. W., Lloyd, R. D. and Marshall, J. H., 1975. "Malignancy risk to humans from total-body gamma-ray irradiation," CONF-730907, pp. 417-428, Proceedings of the 3rd International Congress, International Radiation Protection Association, U.S. Atomic Energy Commission, Oak Ridge, Tennessee.

Mining Enforcement and Safety Administration (MESA) 1975. "Administration of the Federal Metal and Nonmetallic Mine Safety Act, P.L. 89-577, Annual Report to Congress of the Secretary of the Interior."

Modan, B., D. Baidatz, H. Mart, R. Steinitz and S. G. Levin, 1974. Radiation-induced Head and Neck Tumours, Lancet, Vol. 1, p. 7852.

National Academy of Sciences (NAS) 1972. The Effects on Populations of Exposure to Low Levels of Ionizing Radiation, Report of the Advisory Committee on the Biological Effects of Ionizing Radiations, Government Printing Office, Washington, D.C.

National Council on Radiation Protection and Measurements (NCRP) 1975. "Review of the Current State of Radiation Protection Philosophy," Report No. 43, NCRP, Washington, D.C. 20014

Richardson, A. C. B., 1974. The Historical Development of Concepts of Radiation Dose Commitment, Symposium on Population Exposures, CONF-741018, pp. 1-8, National Technical Information Services, Springfield, Virginia 22161.

Rossi, H. H. and A. M. Kellerer 1974. "The validity of risk estimate of leukemia incidence based on Japanese data", Rad. Res. 58, 131

Rowe, W. D. Anatomy of Risk (in press), Wiley, New York, 1977.

Upton, A. C., 1976. Radiological Effects of Low Dose: Implications for Radiological Protection, Rad. Res. (In press.)

U.S. Atomic Energy Commission (AEC) 1974. Draft Generic Environmental Statement - Mix Oxide Fuel, WASH-1327, Vol. 3, Chapter IV, p. J-12, U.S Nuclear Regulatory Commission, Bethesda, Maryland.

U.S. Environmental Protection Agency (EPA) 1973-1. Environmental Analysis of the Uranium Fuel Cycle, Vol. I, p. 11, EPA-520/9-73-003-13, USEPA, Washington, D. C.

EPA, 1973-2, Ibid. Vol. III, p. 12.

EPA, 1973-3, Ibid. Vol II, p. 10.

U.S. Environmental Protection Agency (EPA) 1975. Transportation Accident Risks in the Nuclear Power Industy 1975-2020, EPA-520/3-75-023, Washington, D.C. 20460.

U.S. Environmental Protection Agency (EPA) 1976-1. Reactor Safety Study (WASH-1400), A Review of the Final Report, EPA-520/3-76-009, pp. 2-1 - 2-7, USEPA, Washington, D. C.

U.S. Environmental Protection Agency (EPA) 1976-2. Environmental Radiation Protection Requirements for Normal Operations of Activities in the Uranium Fuel Cycle - Final Environmental Statement, Vol. I, pp. 52-62, EPA-520/4-76-016, USEPA, Washington, D. C. 20460.

EPA 1976-2, Ibid. pp. 187-188

U.S. Nuclear Regulatory Commission (NRC) 1975. Reactor Safety Study, An Assessment of Accident Risks in U.S. Commercial Nuclear Power Plants, Appendix VI, WASH-1400 (NUREG-75/014), National Technical Information Services, Springfield, Virginia 22161.

U. S. Environmental Protection Agency (EPA) 1975. Transportation Accident Risks in the Nuclear Power Industry 1975-2020, EPA-520/3-75-023, Washington, D.C. 20460.